

DOCUMENT RESUME

ED 326 035

EC 232 650

AUTHOR Griesbach, Linda Sue; Polloway, Edward A.
TITLE Fetal Alcohol Syndrome: Research Review and Implications.
PUB DATE 13 Aug 90
NOTE 34p.
PUB TYPE Information Analyses (070) --

EDRS PRICE MF01/PC02 Plus Postage.
DESCRIPTORS *Alcohol Abuse; Alcoholism; *Clinical Diagnosis; *Congenital Impairments; Drinking; *Epidemiology; Handicap Identification; History; Intervention; Mental Retardation; *Neurological Impairments; Pregnancy; *Prenatal Influences; Prevention
IDENTIFIERS *Fetal Alcohol Syndrome

ABSTRACT

Research on fetal alcohol syndrome is reviewed, with particular emphasis on the implications of the syndrome for the development of mental retardation and other handicapping conditions. Attention is given to historical aspects; epidemiology; physiological and behavioral characteristics; and concerns related to diagnosis, prevention, and intervention. The review provides an overview of biological processes and critical periods of gestation, and examines the effects of fetal alcohol syndrome in terms of central nervous system dysfunction, craniofacial malformations, prenatal and postnatal growth retardation, and other less frequent anomalies. The review concludes with a discussion of approaches to prevention and intervention. (42 references) (JDD)

* Reproductions supplied by EDRS are the best that can be made *
* from the original document. *

ED326035

FAS.CS and FAS
8/13/90

**Fetal Alcohol Syndrome:
Research Review and Implications**

Linda Sue Griesbach,
Cecil County (MD) Public Schools

and

Edward A. Polloway
Professor of Education and
Human Development
Lynchburg College

Address correspondence to:
Edward A. Polloway, EdD.
School of Education and Human Development
Lynchburg College
Lynchburg, VA 24501
804-522-8489

Running head: FAS

U.S. DEPARTMENT OF EDUCATION
Office of Educational Research and Improvement
EDUCATIONAL RESOURCES INFORMATION
CENTER (ERIC)

☒ This document has been reproduced as
received from the person or organization
originating it.

☐ Minor changes have been made to improve
reproduction quality.

• Points of view or opinions stated in this docu-
ment do not necessarily represent official
OERI position or policy.

"PERMISSION TO REPRODUCE THIS
MATERIAL HAS BEEN GRANTED BY

Edward A.
Polloway

TO THE EDUCATIONAL RESOURCES
INFORMATION CENTER (ERIC)."

EC 232650

Abstract

Research on fetal alcohol syndrome (FAS) is reviewed with particular emphasis on the implications of the syndrome for the development of mental retardation and other handicapping conditions. Attention is given to historical aspects, epidemiology, physiological and behavioral characteristics, and concerns related to diagnosis, prevention and intervention. Implications of the current research on FAS are discussed.

In recent years, expectant mothers have been made aware of possible dangers they may encounter throughout gestation. Among the most significant are the effects associated with Fetal Alcohol Syndrome (FAS), a condition that has been referred to as the "most frequent known teratogenic cause of mental deficiency in the Western World" (Clarren & Smith, 1978). If such an appraisal of FAS is valid, these effects are truly of great concern and "may ultimately outweigh that of a tragedy even of the magnitude of thalidomide if for no other reason than the sheer number of women child bearing and women of child bearing age who use and misuse alcohol" (Vorhees & Butcher, 1982).

The purpose of this paper is to discuss historical aspects of FAS and epidemiological features, to provide an overview of biological processes and critical periods of gestation and to highlight the characteristics that accompany this disorder. The paper concludes with a discussion of approaches to prevention and intervention.

History

The concern that alcohol is damaging to the developing fetus has been suspected for centuries. From as far back as the Old Testament in the book of Judges 13:7, it is said that in the age of the Philistines an angel appeared to Samson's mother and said, "Behold, thou shalt conceive and bear a son; and now drink no wine or strong drink...".

Cautions were made in the laws of Sparta and Carthage

whereby bridal couples were forbidden to drink wine. The reasoning for this prohibition was to prevent the conception of defective children (Rosman & Oppenheimer, 1985). Later in England note was made of the detrimental effects to the fetus during the "gin epidemic" of 1720-1750. College physicians at the time cited gin to be a leading cause of what they considered to be weak, feeble and distempered children. (Letter to Parliament, January 19, 1725, as cited by Warner & Rosett, 1975).

In 1900 Sullivan, (as cited by Jones & Smith, 1973) reported chronic alcoholic women in Liverpool experienced increased abortion and stillbirth rates, while the surviving offspring exhibited higher rates of occurrence of epilepsy. During the 1920s when prohibition was in effect in the United States there was little interest in the effects of alcohol to the fetus (Rosman & Oppenheimer, 1985). However, reports reappeared in the 1950s especially in France where medical interest was continuing to uncover the injurious effects of alcohol to the unborn (Jones & Smith, 1973). Jones, Smith, Ulleland and Streissguth (1973) coined the term "fetal alcohol syndrome" after studies of 8 unrelated offspring born to chronically alcoholic mothers showed a pattern of growth and morphogenesis. FAS was described as a recognizable pattern of major and minor malformations, growth deficiency and developmental disability arising from maternal alcohol consumption during pregnancy (Smith & Jones, 1973).

These historical reports indicate that the adverse effects of alcohol have been considered for many years by various

individuals. However, it has only been relatively recently that efforts have been made to accurately describe these effects and work toward preventive interventions (Delaney & Hayden, 1977).

Epidemiology

It has proven difficult for researchers and public health professionals to estimate the exact prevalence of fetal alcohol syndrome. Based on the Sixth Annual Report to the U.S. Congress on Alcohol and Health from the Secretary of Health & Human Services, the best estimates of occurrence seem to be between 1 to 3 cases per 1,000 births (Warren & Bast, 1988). Other studies have also reinforced this approximate rate of occurrence, placing figures at 1 in 650 births (Webb, Hochberg, & Sher, 1988). At this rate, FAS would be the leading known cause of mental retardation, even ranking ahead of Down Syndrome (Abel & Sokol, 1986), commonly referred to as the most prevalent cause. Other data bases however tend to question the validity of that assumption and rather categorize FAS as the third leading cause of mental retardation (Ouellette, 1984; Streissguth & La Due, 1987). However, there is little question that FAS is one of the leading causes of mental retardation in the United States.

FAS has also been reported in many other nations as well. Cases have been diagnosed in Australia, Belgium, Brazil, Canada, Chile, Czechoslovakia, France, Germany, Hungary, Ireland, Italy, Japan, South Africa, Spain, Sweden and Switzerland (Abel & Sokol, 1986). Other sources have documented FAS as occurring worldwide from the Soviet Union to New Zealand (Benderly, 1989).

FAS is the term used to describe the full-blown effects caused by heavy drinking; as such, its accurate use requires specific criteria for diagnosis. However, all women who drink heavily throughout pregnancy do not give birth to infants with Fetal Alcohol Syndrome. Full-blown FAS occurs in 40% of the cases (Streissguth, Landesman-Dwyer, Martin, & Smith, 1980) while many more may experience a less severe form referred to as Fetal Alcohol Effect (FAE). FAE may include any of the features seen in FAS (as elaborated on below) and may also be associated with failure to thrive, attention deficit disorders, and learning and developmental disabilities. Included in these effects are a variety of psychosocial, behavioral and neurological disorders such as hyperactivity that can be linked to maternal alcohol consumption as well. To get a better understanding of the number of occurrences, there were 3,704,000 babies born in the U.S. in 1982. Full FAS was estimated to occur in 3,700 to 7,400 babies with the frequency of FAE almost triple that, with estimates being from 11,100 to 18,500 babies in that year (Smith, Jones, & Hanson 1976).

Another term that refers to this type of birth defect is "alcohol-related birth defects" (ARBD). ARBD includes any birth defects or complications during pregnancy that are attributed to alcohol (Warren & Bast, 1988).

Pregnancies may also end as a result of alcohol-related embrotoxic effects such as abortion and stillbirth. Reports from the University of Washington showed that, among mothers with a

history of alcoholism, 17% of their offspring were stillbirths or died within the first week of life, 44% showed some form of mental retardation while 32% had abnormal physical features (Streissguth & La Due, 1987).

Specific Problems in Native American Communities

While signs of FAS have been identified in children of virtually all ethnic backgrounds, large variations in the prevalence of FAS have been observed in different populations. For example, while FAS may average 1 in 750 births in many populations, other areas have yielded estimates as high as 1 in 100 births, the incidence rate cited on some Indian Reservations (Streissguth & La Due, 1987). Perhaps these data should not be viewed as surprising given that the alcoholism rate in the population is about 5 times greater than for the general population (The fetal alcohol syndrome public awareness campaign, 1979). For this reason, special attention is given to this particular population.

There are many problem areas that can be directly linked to alcohol. In Native American communities in Canada, the government spends "upwards of 500 million dollars a year" in various areas in efforts to alleviate the problems of alcohol and drugs in their native communities (The Indian health promotion..., 1985, p.10). The problems of FAS and FAE are so great that "preliminary estimates indicate that as many as half of all native children now in school may be victims of this phenomenon" (The Indian health promotion..., 1985, pg.14).

Unfortunately, it seems that many of these children are affected both prior to and after birth by the neglect and abuse associated with alcoholism (Four Worlds Project, 1984).

A report prepared by the Department of Indian Affairs and Northern Development highlighted the following:

- * between 50 & 60 percent of Indian illnesses and deaths are alcohol-related.
- * the current rate of prenatal and neonatal mortality is estimated to be 60 percent higher than the national rate.
- * the proportion of children in foster care is approximately 5 times higher than the national rate (The Indian health promotion..., 1985).

FAS and FAE in individuals who are Native Americans are well-illustrated in Dorris' "The Broken Cord" (1989), a personal account by the author about his adopted Sioux Indian baby. The boy was diagnosed as being mentally retarded but later was identified as having severe learning disabilities. Eventually these problems were linked to the fact that Adam's biological mother had been an alcoholic and had died of alcohol poisoning. Adam was born 7 weeks premature and was consistently late in meeting developmental milestones. Dorris (1989) documents the various problems experienced by Adam including severe epileptic seizures (up to nine times a day), overimpulsivity, inappropriate social behavior, and deficits in abstract thought.

Biological Processes of FAS

Unfortunately, many mothers don't realize that when they drink their unborn child drinks as well. Alcohol passes from the bloodstream of the mother through the placenta and into the bloodstream of the child. Alcohol passes rapidly from the maternal circulation to the fetus in approximately the same concentration. Alcohol levels in the fetus tend to fall more slowly than in maternal circulation. Consequently alcohol is often detectable in the fetus long after it has cleared the mother's circulation and remains in the amniotic fluid for several hours after ingestion. Even a small amount of alcohol can have serious effects on the development of the fetus. Due to the fact that alcohol is a depressant, it often decreases growth rate, affects the overall brain development and causes other abnormalities in the fetus (Ouellette, 1984).

Safe levels of alcohol intake during pregnancy, if any, have not been determined. As a result, Ouellette (1984) suggested total abstinence from drinking from conception throughout the entire pregnancy. Estimates have been made that 10 percent of birth defects caused by alcohol are associated with as little as 2 drinks a day (Seixas & Youcha, 1985). This is reiterated again by Light (1988) who states that "even one or 2 drinks at any time during pregnancy may have irreversible adverse effects on the developing fetus" (p.88). It is generally recognized, however, that the first trimester is the most critical period in this situation, particularly, the 4th to 10th weeks' of gestation. During this period cells are rapidly dividing, forming the vital

organs and the structure of the brain. The time of conception is particularly significant and yet problematic since many women would not yet be aware of the fertility (Ernhardt, Sokol, Martier, Moron, Nadler, Ager & Wolf, 1987). It is likely that heavy drinking at this stage has the greatest effect on fetal maldevelopment whereas heavy alcohol consumption near term may have a greater effect on fetal nutrition and size (Ouellette, Rosett, Rosman & Weiner, 1977) since it is during the 2nd and 3rd trimesters that the fetus is growing the most overall and thus there is an increase in tissue growth and organ size. Brain cells are also maturing through the third trimester. As a result alcohol-related disruption in the amniotic fluids have been shown to be related to continued maternal drinking in the final trimester as well (Ernhardt et al., 1987).

Ernhardt and colleagues (1987) conducted a study specifically to determine the most critical period of gestation. They found 359 neonates who had been prenatally exposed to alcohol. These infants were all offspring of disadvantaged urban women located by the Michigan Alcoholism Screening Test, a questionnaire used in detecting chronic alcohol problems. The study concluded that the consumption of 3 ounces of absolute alcohol per day (equal to 6 cans of beer, glasses of wine or mixed drinks), at or about the time of conception results in a significant increase in the risk of alcohol teratogenicity. This study generated strong recommendations for those women planning pregnancy to avoid alcohol consumption totally or at the very

least to reduce consumption to a minimal level in order to avoid alcohol-related abnormalities. While there is limited evidence concerning the precise ~~timing~~ and levels of alcohol consumption in women, there is even more ambiguity concerning the effects of excessive alcohol use in males. Correlations have been made between alcohol abuse and infertility in men (Finn & O'Gorman, 1981). It has also been suggested that such long term consumption can reduce sperm motility and abnormalities ultimately resulting in loss of sperm all together. The unresolved question however, is whether or not a child conceived during a period of paternal heavy drinking will exhibit alcohol related birth defects (Finn & O'Gorman, 1981). It has been postulated by some physicians that this type of heavy drinking may damage sperm and induce miscarriages (Seixas & Youcher, 1985).

Characteristics

FAS is defined as a pattern of congenital malformations associated with fetal exposure to alcohol (Johnson & Scruggs, 1981). The characteristics of the syndrome can be separated into three primary features: central nervous system dysfunction, craniofacial malformations, and prenatal and postnatal growth development. A diagnosis of FAS is warranted when a child has a cluster of disorders within these three areas.

CNS Dysfunction

The effects of alcohol on the central nervous system may be initially seen through the presence of mild to moderate

retardation. According to two reports, the average IQ for children with FAS is approximately 68 although the range is extremely wide (Johnson & Scruggs, 1981; Chasnoff, 1983).

Streissguth, Herman, and Smith (1978) at the University of Washington studied the development of 20 children of chronically alcoholic mothers. Standardized, age appropriate psychological tests were administered on an individualized basis with the results indicating intellectual levels ranging from an IQ of 15 in a child with moderate/severe fetal alcohol syndrome to an IQ of 105 in a mild case. The mean intellectual level for all 20 patients was 65, thus averaging in the mildly retarded range.

Some FAS children have been noted to have average intelligence. As the child grows older however, it is common to see degrees of hyperactivity, impulsivity, short attention span, distractibility, lack of inhibition, overfriendliness, overinquisitiveness, poor social judgment, poor sensitivity to social cues and excessive demands for attention (Streissguth & La Due, 1987).

An additional effect alcohol may have on the central nervous system is microcephaly. This may also be considered a craniofacial disorder. Generally, it is of prenatal onset although occasionally it becomes more apparent with time. This condition reflects deficient brain growth but, as neurological and psychological studies demonstrate, normal brain size does not necessarily predict normal brain structure or function after intrauterine alcohol exposure (Clarren & Smith, 1978).

Fetal alcohol syndrome newborns often suffer symptoms of alcohol toxicity as would a chronic alcoholic. In fact, there have been reports of babies literally "born drunk". The mother uses alcohol as a means of killing pain at the onset of labor and since the mother and child are joined until the umbilical cord is severed they continue to share the same blood alcohol level as well. These babies need to be "detoxified" as they come into the world and apparently go through some of the same physiological trauma that an adult would encounter as their blood alcohol level drops suddenly (Seixas & Youcha, 1985).

Other symptoms such as tension, tremors, and anxiety seizures are also frequently displayed soon after birth. It is most common that these symptoms manifest themselves in the first 24 hours, usually appearing within 6-12 hours of birth and lasting approximately 7 days (Elliot & Johnson, 1983). Epileptic seizures may also be found and are often extremely serious. Although they occur early on and are seen less frequently beyond the neonatal period, some sources report finding that these symptoms persist through the early school years (Erbenbach, 1979).

Infants born to alcoholics or even heavy drinkers have been noted to be more restless during sleep as well as not sleeping as much as other children overall (Rosett & Sander, 1979). Abnormal electroencephalographic (EEG) activity has been apparent for as long as six weeks after birth for these infants.

Other CNS symptoms include jitteriness, opisthotonos

(arching of the back), hyperacusis (hypersensitivity to loudness), either hypotonia or increased muscle tone, and an overall high degree of irritability by comparison to drugfree infants (Streissguth, 1986). Delayed reaction time as well as decrements in fine and gross motor performance are also evident in many cases (Warren & Bast, 1988).

Finally, alcohol may affect the CNS through speech and language disorders. This can be seen through slurring, difficulty articulating as well as decrements in overall spoken language and verbal comprehension. In a study done at the University of Colorado, there was high incidence of speech and language disorders prevalent in a majority of 14 FAS patients tested. 93% of those tested showed signs of receptive and expressive language delays. Speech and language delays ranged from a one and a half year delay for a six year old, a 4-6 year delay for a eleven-year-old and completely unintelligible speech was noted in a twelve-year-old child. Three other children exhibited other speech defects while another showed signs of echolalia (Church & Gerkin, 1988).

Craniofacial Malformations

The cause of these speech impediments could also be attributed to a second area of FAS features, craniofacial malformations. In order to be diagnosed with FAS, there must be at least two or more of these craniofacial defects. Those defects that may result in speech problems could be a result of skeletal and growth deformalities of the jaws, micrognathia

(small chin), or as a result of a cleft plate which is also fairly common with this syndrome. Other frequently observed facial features include a low nasal bridge, a short upturned nose, indistinct philtrum (the ridges running between the nose and the mouth), short palpebral (eye slits) fissures, a short depressed midface, a thin upper lip, epicanthal folds, bilateral ptosis (drooping eyelids), strabismus (crossed eyes), microcephaly (as noted earlier) and in some cases, malformations of the external ear (Church & Gerkin, 1988; Streissguth & La Due, 1987). Erbenbach (1979) considers these features to be as distinctive to FAS as the classic stigmata are to Down Syndrome.

Prenatal and Postnatal Growth Development

A third major manifestation area required for diagnosis of FAS is having prenatal or postnatal growth retardation (below the 10th percentile). In general children with FAS remain more than 2 standard deviations below the mean for height and weight with weight being more severely limited. The low weight of these children is a result of decreased adipose tissue which is also a consistent feature seen in FAS victims (Erbenbach, 1979). This characteristic of lower birth weight is the most reliably observed effect attributed to utero exposure. Low birth weight is widely recognized as a sign of risk of infant mortality and long term abnormalities in neurological development and intelligence (Ouellette et. al., 1977).

Other Characteristics

While CNS dysfunction, craniofacial malformations and pre-

and postnatal growth abnormalities are the most common within this syndrome as well as also being the criteria for diagnosis, they are not the only characteristics that have been noted. Some of the other nonspecific anomalies that are seen in conjunction with FAS include eye and ear defects. These types of anomalies are often associated with a number of neonatal risk factors including hyperbilirubinemia, low birth weight and possible antibiotic treatment. Risks are also more prevalent in children with craniofacial features. Common ocular disorders include strabismus, myopia, perception disorders and astigmatisms. High incidence of sensorineural hearing impairments and serous otitis media problems can often be linked to fetal alcohol syndrome. These disorders may frequently cause the commonly seen speech and language disorders (Church & Gerkin, 1988).

Some of the other anomalies that may be identified are heart murmurs, aortic stenosis, hemangiomas (a type of birthmark), fingerprint and palmar crease abnormalities, club foot, bone fragility, Hutchinson's teeth, limb reduction, and congenital dislocation of joints (Church & Gerkin, 1988). Other nonspecific features documented in FAS cases may include an increase in respiratory problems, the most frequently cited of which is recurrent upper respiratory tract infections. Studies done by Jones and Smith (1973) looked at 3 specific cases of FAS infants all of whom were Native American Indians. Through their study they found a frequency of chronic upper airway obstructions caused complication in growth due to the increased work of

breathing as well as feeding difficulties. It is increasingly important to be aware of these defects as there is an increased risk of sudden infant death syndrome and pulmonary hypertension (Usowicz, Golabi, & Curry, 1986).

Due to these varied possible features, it is essential that each case be considered individually, putting the child's safety as top priority (Webb, Hochberg, & Sher, 1988). Consequently, FAS may be overlooked unless these clusters of subtle characteristics are viewed by a trained eye (Streissguth & La Due, 1987). This provides some explanation as to why there are so many difficulties in diagnosing this multifaceted syndrome.

Diagnosis

Being aware of the presence of FAS is not always easy since there are varied factors to be considered. A study done by Sokol and colleagues to identify alcohol abuse in prenatal clinics found that newborns were being misdiagnosed in at least three of every four alcohol-abusing patients (Sokol, Martier, & Ernhardt, 1982). Additional difficulty comes into play as few patients limit their substance abuse to just alcohol (Chasnoff, 1988).

Another obstacle associated with the diagnosis of this syndrome is that many of these characteristics are not apparent for several years. Graham, Hanson, Darby, Barn and Streissguth (1988) reported on a study conducted on two groups of 4 year old children that had been examined by a dysmorphologist who had no previous knowledge of examination results or prenatal exposure and categorization of fetal alcohol effects. These groups were

divided based on their mothers' self-report of drinking during pregnancy. Children born to 108 mothers were classified in a heavy drinking group. The criteria for this group were mothers who reported drinking equal to or greater than 1 ounce of alcohol per day during pregnancy or 45 or more drinks per month. The comparison group was 97 mothers reported drinking never or infrequently, which is 6 drinks or less per month during pregnancy and had no previous record of intoxications or binges. These groups were matched according to the following variables: maternal race, age, parity, education, socioeconomic status, marital status and smoking. Comparison of these two groups was done at birth. The assessment included size at birth, notation of any dysmorphic features and overall judgement as to any other signs of fetal alcohol syndrome. As mentioned above, these children were individually judged again at 4 years of age. Growth data from birth, 8 and 18 months and 4 years were all compared with standardized growth curves. A checklist was used to rate alterations in morphogenesis that had been reported in children with fetal alcohol effects (Graham et al., 1988).

The results of the children in the maternal heavy drinking group showed 20.4% with fetal alcohol effects at age four versus 9.3% from the comparison group. A total of 80% of children judged to have FAE at birth were also found to be affected at 4 years of age. More intriguing however, only 50% of those identified as having fetal alcohol effects at 4 years were identified at birth. This study indicates that children

suspected to be at risk of FAE should be monitored at birth and during the preschool years. This evidence is crucial in diagnosing FAS since the "absence of symptoms and signs at birth is not necessarily predictive of the absence of an effect" (Graham et al., 1988, pg.777).

FAS has been diagnosed in infants, children, adolescents and even adults. In the newborn, the diagnosis is often made from facial characteristics and growth deficiency while functional deficits often elicit clues as the child matures (Jones & Smith, 1973). As a result, Graham and colleagues (1988) suggest that while it might be possible to identify alcohol-affected-children using morphology and size alone, diagnosis can become more accurate if behavioral data are collected on older children particularly in the areas of distractibility and hyperactivity.

Certainly the earliest diagnosis of FAS or FAE is preferable. In a study conducted in Helsinki, efforts were made to predict FAS and/or FAE prenatally. This was the first attempt at this early diagnosis due to the difficulties in performing a prospective study on drinking women, who often neglect prenatal care. This evaluation looked at usefulness of alpha-fetaprotein, human placental lactogen and pregnancy-specific, beta₁-glycoprotein in the prenatal diagnosis of fetal alcohol syndrome (Halmesmaki, Autti, Granstrom, Heiteinhoimo, Raivio, & Ylikorkala, 1986). Alpha-fetaprotein is synthesized in the fetal liver and can be found in high concentration in the fetal circulation and amniotic fluid.

This particular study considered 35 women who drank throughout pregnancy. All patients drank at least 100 gm of ethanol weekly, and some consumed up to 2 bottles of vodka per day during the first half of pregnancy. There were also 19 abstinent women considered in this test. Thirty-seven percent of these 42 patients gave birth to infants with FAS, one of whom was stillborn. All of the infants born to the abstinent women were born healthy (Halmesmaki et al., 1986). Venous blood samples were used to consider both the ranges for alpha-fetaprotein and beta₁-glycoprotein. Seventy-four percent of the alpha-fetaprotein concentrations in drinkers with healthy infants fell within the normal range. The majority of subjects with FAS showed low alpha-fetaprotein. The levels of human placental lactogen did not predict FAS however. There were also subnormal levels of pregnancy-specific beta₁-glycoprotein. Thus these two protein levels proved to behave differently in the women whose infants were diagnosed with FAS. As a result this information may reflect primary or secondary effects of ethanol abuse in pregnancy and allow measures to be taken to predict and prevent FAS (Halmesmaki et al., 1986).

There are a variety of other techniques used to predict possible structural malformations or complications in pregnancy which may be linked to alcohol consumption. The most frequently used assessments are ultrasound and amniocentesis which are often diagnostic tools used in conjunction with one another. These techniques can detect such FAS characteristics as microcephaly,

limb defects and cardiac abnormalities (Ouellette, 1984).

Prevention and Intervention

Since FAS is potentially the most preventable form of mental retardation, it is important to consider all prevention and intervention possibilities that can mitigate against this syndrome. This is why it is critical that identification of women who may be at high risk of alcohol abuse and their children who may show signs of FAS or FAE needs to be done as early as possible.

There have been efforts made to reach the public through awareness activities designed to reduce maternal drinking. Another mode to provide prevention and intervention is through means such as screening and counseling during the pre- and post natal periods. This second form of intervention is more difficult as it requires everyone that is directly or indirectly involved in the gestation period to be educated and trained in detecting possible signs of alcoholism before birth as well as the symptoms associated with FAS and FAE after birth (Baumeister & Hamlett, 1986). These efforts are crucial because a woman automatically increases her chances of having a healthy baby as soon as her drinking ceases. Yet, for any kind of health promotion campaign to achieve success the help and participation of all health professions is vital (Program Strategies, 1987).

In the areas of screening and counseling, there are various programs that can be considered. The most desirable time for screening and preventative counseling to occur is prior to

pregnancy. Programs have been implemented by local organizations, by state and county councils on alcoholism, and by mental health departments. Whatever the intervention or prevention method may be, whether it be in the form of education, counseling or treatment, the concern should not be restricted to women in the prenatal setting alone. While efforts to increase public awareness can obviously be given to clients receiving gynecologic examinations, pregnancy tests, and those that attend family planning meetings (Stephens, 1981) that is not the only area to provide public awareness. Additional information can be elicited through the staffs of neonatal nurseries, pediatric units, pediatricians, family practitioners and early intervention programs (Ouellette, 1984). The efforts of other less traditional areas can often be just as beneficial; these may include pharmacies, marriage license bureaus, maternity clothes stores, supermarkets, and laundromats (Program Strategies, 1981).

As forms of public awareness, educational campaigns, media coverage, professional training and volunteer services such as crisis telephone hotlines are all presently being utilized (Screissguth & La Due, 1987). One of the most effective means of public awareness, put into effect as of November 1989, requires liquor stores and bars to post warnings linking the tragedies of birth defects to alcohol consumption (Waldman, 1989). Additionally federal law also requires that all alcoholic beverages carry a warning about possible birth defects and other drinking hazards as well.

Federal involvement can also have a tremendous effect on public interest. For instance, in 1984 then President Reagan declared January 15-21 National Fetal Alcohol Syndrome Awareness Week. This initiated federal funding toward intensive treatment centers for alcoholic women (Baumeister & Hamlett, 1986).

Several studies have been conducted to estimate the cost of various intervention and prevention programs. It is difficult to calculate exactly what percentage of funding is allotted specifically for FAS prevention and intervention. These estimates do however reflect optimistic steps forward. This is evident when considering that Little, Young, Streissguth and Uhl (1984) projected cost effectiveness to be about \$1 million which would be approximately equal to the cost of lifetime maintenance for one person who is severely retarded and dependent (Streissguth & La Due, 1987).

Baumeister and Hamlett (1986) also conducted a survey to determine the number of states that provide prevention services for FAS. In addition, this survey gathered data on the overall incidence in each state. Thirty-nine states responded to the questionnaires mailed to the primary health agency. 49% of the respondents found FAS to be a major health problem in their state. The highest incidence was reported in South Dakota which is the only state in which birth certificates specifically require notation of any diagnosis of the syndrome.

In considering the prevention organization, 71% of the

correspondents listed more than one organization within that state that has prevention programs for FAS. The March of Dimes was typically the most avid supporter. About half of the states were currently conducting public awareness activities which seems to be a positive step forward. It is difficult to collect reliable data in this area but these figures illustrate that efforts are being made to prevent FAS and FAE. The annual funding reported from these reports ranged from \$20,000-\$200,000. However, most FAS prevention efforts are included among other staff activities as this type of prevention does not occur frequently enough to be regarded as a specific budget item (Baumeister & Hamlett, 1986).

A study conducted by the Research Triangle Institute for Alcohol, Drug Abuse, and Mental Health Administration illustrates a breakdown of costs. Health treatment costs were estimated at \$14.9 million for 3,600 babies born with FAS during the year of 1980. Within the same year \$670 million was spent on 68,000 FAS children under the age of 18 while \$760 million was estimated for 160,000 FAS adults. If the estimates of the rate of occurrence were correct as cited as approximately 1 in 600, the direct service cost per year for FAS individuals as a whole would be approximately \$2.4 billion each year (Program Strategies, 1987).

While the ultimate goal of the time, effort and funding involved in these programs is to prevent FAS entirely, intervention programs are making great strides as well. This is particularly visible in one case study reported by Barbour

(1989). The subject was born at 4 pounds 14 ounces and displayed many of the characteristics indicative of FAS including: microcephaly, short palpebral fissures, epicanthal folds, ptosis, a flattened nasal bridge, maxillary, hypoplasia, an indistinct philtrum, a thin upper lip and a small mandible. In addition, it was later discovered that he suffered from congestive heart failure as well as tremors, irritability, and hypersensitivity to sound and was far more difficult to console than a non-affected infant. Additionally the infant exhibited overall disinterest in food allowing little chance for normal development as his nutritional needs were not being met. However, despite his developmental delays, he was reported as progressing steadily and was mainstreamed into a regular kindergarten. As Barbour noted, since FAS is a "relatively new syndrome," the possible outcomes are not yet known. Thus, it appears quite likely that FAS children could greatly benefit from early intervention programs. Her advice is "it is essential that we identify the special needs of children with fetal alcohol syndrome as well as the intervention programs that will best meet them" (Barbour, 1989, pg.46). Several such programs have already been initiated in the United States and in Europe.

Legal Responses

When prevention techniques have not shown any decrease in occurrence, there have been some cases where legal provisions have been deemed to be warranted. In February of 1990, a Wyoming judge dismissed child abuse charges against a pregnant 29 year

old woman who had been charged with child abuse for being intoxicated during pregnancy. Prosecutors nationwide are extremely upset by the serious harm being done to babies and are putting child abuse and drug laws to new use. However, civil liberties groups view such remedies as unconstitutional and see it much more feasible to provide early treatment rather than punishment. Nevertheless there have been about 35 nationwide cases where women have faced criminal charges for drugs and alcohol use while pregnant. Most charges have ultimately been dismissed as doctors and lawyers acknowledge a fine line as to the mother's legal obligation. Many cases revolve around the issue that the fetus has separate interests from the mother and, while a mother has the legal right to abort a fetus, that does not entitle her to completely disregard prenatal care for the unborn child (Lewin, 1990).

Conclusion

The goal of this paper has been to discuss the nature of fetal alcohol syndrome and fetal alcohol effects. Alcohol has been suspected to be a teratogenic cause of birth defects for centuries, yet this knowledge has not been emphasized sufficiently until quite recently. This is apparent since FAS remains one of the three leading causes of mental retardation, affecting between 1 and 3 infants for every 1,000 births.

What makes it even more critical that this syndrome be brought to public knowledge is the fact that no one precisely

knows when or how much alcohol it would take to harm the fetus. Streissguth stated in a 1990 public media presentation, "there is no safe time and there's no safe amount". Additionally, the problem of FAS is a universal one. The syndrome has been diagnosed in women of all age groups, racial and socioeconomic backgrounds imbibing all types of alcohol. Some populations seem to be more prevalent than others however as seen in many Native American communities.

The effects of FAS and FAE can be seen in a variety of forms such as central nervous system dysfunction, craniofacial malformations, pre- and postnatal growth development as well as in several other less frequent anomalies. The vast number of FAS traits are frequently overlooked however and can go undiagnosed for many years causing the affected individuals and their families tremendous frustration. For this reason, it is critical that special and regular education personnel and other human services professionals become better informed and thus be able to assist in action on what is arguably the most preventable disorder. Additionally, as Delaney and Hayden (1977, p.157) noted "until total preventative measures can be successful to eliminate FAS..., interventions for FAS infants must be developed to provide these potentially severely handicapped infants with the opportunity to live as near-normal lives as possible."

Acknowledgements

The senior author would like to acknowledge the assistance of J. David Smith and Merrill P. Tolbert on an earlier draft of this manuscript.

REFERENCES

- Abel, E.L., & Sokol, R.J., (1986). Fetal alcohol is now a leading cause of mental retardation. Lancet, 8517, 1222.
- Barbour, B.G. (1989). Is fetal alcohol syndrome completely irreversible? MCN, 14 (1), 44-46.
- Baumeister, A.A., & Hamlett, C.L. (1986). A national survey of state sponsored programs to prevent fetal alcohol syndrome. Mental Retardation, 24, 169-173.
- Benderly, B.L., (1989). Saving the children. Health, 21, 74.
- Chasnoff, I.J., (1988). Drug use in pregnancy parameters of risk. The Pediatric Clinic of North America, 35, 1403-1411.
- Church, M.W., & Gerkin, K.P. (1988). Hearing disorders in children with fetal alcohol syndrome: Findings from case reports. Pediatrics, 82, 147-154.
- Clarren, S.K., & Smith, D.W. (1978). The fetal alcohol syndrome. New England Journal of Medicine, 298, 1063-1067.
- Delaney, S., & Hayden, A. (1977). Fetal alcohol syndrome: A review. AAESPH Review, 2, 164-168.
- Dorris, M. (1989). The broken cord. New York: Harper & Row.
- Elliot, D.J. & Johnson, N. (1983). Fetal alcohol syndrome: Implications and counseling considerations. Personnel and Guidance Journal, 62, 67-69.
- Erbenbach, W.J. (ED). (1979). Guidelines for pupil services. Wisconsin State Department of Public Instruction. (ERIC Document Reproduction Service No. ED 186 826).

Ernhardt, C.B., Sokol, R.J., Martier, S., Moron, P., Nadler, D., Ager, J.W. & Wolf, A. (1987). Alcohol teratogenicity in the human: A detailed assessment of specificity, critical period, and threshold. American Journal of Obstetrics and Gynecology, 156, 33-39.

Finn, P., & O'Gorman, P.A. (1981). Teaching about alcohol, concepts, methods and classroom activities, Boston, MA: Allyn & Bacon.

Four Worlds Development Project. (1984). Understanding and preventing the problem of alcohol and drug abuse. Alberta, Canada: Lethbridge University, Adult Education Services.

Graham, M.W., Hanson, J.W., Darby, B.L., Barr, H.M., & Streissguth, A.P. (1988). Independent dysmorphology evaluations at birth and 4 years of age for children exposed to varying amounts of alcohol in utero. Pediatrics, 81, 772-778.

Halmesmaki, E., Autti, I., Granstrom, M.L., Heiikinho, M., Raivio, K.O., & Ylikorkala, O., (1986). Alpha-fetoprotein, human placenta lactogen, and pregnancy-specific B₁-glycoprotein in pregnant women who drink: Relation to fetal alcohol syndrome. American Journal of Obstetrics and Gynecology, 155, 598-601.

Johnson, M.J., & Scruggs, T.E. (Ed.). (1981). All our children: Handicapped and normal. Albuquerque, NM: Paper presented at the National Indian Child Conference. (ERIC Document Reproduction Service NO. ED 217674).

Jones, K.L., & Smith, D.W. (1973). Recognition of the fetal alcohol syndrome in early infancy. The Lancet, 7835, 999-1001.

Jones, K.L., Smith, D.W., Ulleland, C.N., & Streissguth, A.P. (1973). Patterns of malformation in offspring of chronic alcoholic mothers. The Lancet, 7815, 1267-1271.

Lewin, T., (Feb. 5, 1990). Drug use in pregnancy: New issue for the courts. New York Times, A14.

Light, W.H. (1988). Alcoholism & women, genetics and fetal development. Springfield, IL: Charles C. Thomas.

Little, R.A., Young A., Streissguth A.P., & Uhl, C.N. (1984). Preventing fetal alcohol effects: Effectiveness of a demonstration project. In Ciba Foundation Symposium (105), Mechanisms of alcohol damage in utero. London: Pitman.

Ouellette, E.M., Rosett, H.L., Rosman, N.P., & Weiner, L., (1977). Adverse effects on offspring of maternal alcohol abuse during pregnancy. The New England Journal of Medicine, 297, 528-530.

Ouellette, E.M., (1984). Developmental handicaps: Prevention and treatment, II. A cooperative project between University affiliated facilities and state MCH/CC programs. Rockville, MD: Office for Maternal and Child Health Services. (ERIC Document Reproduction Service No. ED 276 193).

Program strategies for preventing fetal alcohol syndrome and alcohol-related birth defects. (1987). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Rosman, N.P., & Oppenheimer, E.Y. (1985). Maternal drinking and the fetal alcohol syndrome. In S. Harel, & N.J. Anastasiow (Eds.), The at-risk infant: Psycho/socio/medical aspects. (pp.121-126). Baltimore, MD: Paul H. Brookes.

Seixas, J.S., & Youcha G. (1985). Children of alcoholism: A survivor's manual. New York: Crown Publishers.

Smith, D.W., Jones K.L., & Hanson, J.W., (1976). Perspectives on the cause and frequency of FAS. Annals of the New York Academy of Science, 273, 140-145.

Sokol, R.J., Martier, S. & Ernhardt C., (1982). Identification of alcohol abuse in the prenatal care clinic. National Institute on Alcohol abuse and alcoholism. Washington D.C.: U.S. Government Printing Office.

Stephens, C.J. (1981). The fetal alcohol syndrome: Cause for concern. American Journal of Maternal Child Nursing, 6, 6.

- Streissguth, A.P. (1986). The behavioral teratology of alcohol: Performance, behavioral, and intellectual deficits in prenatally exposed children. In J.R. West (Ed.), Alcohol and brain development (pp. 3-8). New York, Oxford: Oxford University Press.
- Streissguth, A.P., Herman, C.S., & Smith, D.W., (1978). Stability of intelligence in FAS: A primary report. Alcoholism Clinical Experimental Research, 2, 165-171.
- Streissguth, A.P. & La Due, R.A. (1987). Fetal alcohol: Teratogenic causes of developmental disabilities. In S.R. Schroeder (Ed.), Toxic substances and mental retardation: Neurobehavioral toxicology and teratology. Washington D.C.: American Association on Mental Deficiency.
- Streissguth, A.P., Landesman-Dwyer, S., Martin, J.C., & Smith, D.A. (1980). Teratogenic effects of alcohol in humans and laboratory animals. Science, 209, 353-361.
- The Fetal Alcohol Syndrome Public Awareness Campaign. (1979). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism. (ERIC Document Reproduction Service No. ED 179 287).
- The Indian health promotion and disease prevention act. (1985). Hearing before select committee on Indian affairs. United States Senate (99th Congress).
- Usowicz, A.G., Golabi, M., & Curry, C. (1986). Upper airway obstruction in infants with fetal alcohol syndrome. American Journal of Diseases of Children, 140, 1039-1041.
- Vorhees, C.V., & Butcher, R.E. (1982). Behavioral teratogenicity. In K. Snell (Ed.), Developmental toxicology (pp. 274-293). New York: Praeger.
- Waldman, H.B. (1989). Fetal alcohol syndrome and the realities of our time. Journal of Dentistry for Children, 56, 435-437.

Warner, R.H., & Rosett, H.L. (1975). The effects of drinking on offspring: An historical survey of the American and British literature. Journal of studies on Alcohol, 36, 1395-1420.

Warren, K.R., & Bast, R.J. (1988). Alcohol-related birth defects: An update. Public Health Reports, 103, 638-642.

Webb, S., Hochberg, M.S., & Sher, M.R. (1987). Fetal alcohol syndrome: Report of a case. Journal of American Dental Association, 116, 196-198.